Sleep homeostasis and brain temperature

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SUMMARY

Interactions between sleep and wakefulness, EEG slow-wave activity (SWA) during non-rapid-eye-movement sleep (NREMS), and cortical temperature (T_{CRT}) were investigated in the rat under different experimental conditions.

Slow oscillations (< 4 Hz) are a dominant feature of the EEG during NREMS in all mammalian species. According to the two-process model of sleep regulation, power density in this frequency range (i.e. SWA) reflects a homeostatically regulated process and is a measure of sleep intensity. The model postulates that the level of SWA depends solely on the prior history of sleep and wakefulness and predicts that SWA at the onset of NREMS increases with increasing duration of prior wakefulness and decreases during NREMS according to exponential functions.

A first aim of the present study was to test whether in the rat the level of SWA is determined by the sequence of the vigilance states. The parameters of the exponential functions describing the increase and decrease of the process underlying these variations in SWA were determined in a four-day experiment during which rats were sleep deprived on the second day. Computer simulations revealed that the time course of SWA could be simulated on the basis of the distribution of the vigilance states during baseline as well as during recovery from 24 h and 12 h of sleep deprivation (Chapters 4,5).

The regulation of sleep and thermoregulation are intimately related. Brain temperature typically decreases in NREMS in many mammalian species. Based on this and other observations it has been hypothesized that NREMS, and in particular NREMS of high intensity, serves a thermoregulatory function in that it cools the brain. The relationships between brain temperature, vigilance states and SWA were investigated during baseline, and after 12 h and 24 h of sleep deprivation. Furthermore this relationship was investigated after a challenge of the thermoregulatory system by a 12-h exposure to a low ambient temperature (4°C).

The correlations between T_{CRT}, vigilance states, and SWA were calculated. Variations in T_{CRT} were highly, negatively correlated with variations in the

duration of NREMS (Chapters 2,6). In contrast no significant correlation was found between SWA and T_{CRT} during both baseline and during recovery from sleep deprivation; the rate of the decrease of T_{CRT} in NREMS episodes was not related to the level of SWA (Chapters 2,3). In order to investigate to what extent the sequence of the vigilance states determined T_{CRT}, its time course was simulated by assuming that T_{CRT} increases during waking and REMS, and decreases in NREMS according to exponential functions (Chapter 6). The simulation showed that the time course of T_{CRT} could be accurately predicted on the basis of the vigilance states with an additional low-amplitude endogenous circadian modulation of T_{CRT}. Since T_{CRT} and SWA did not correlate and because T_{CRT} could be simulated without taking the level of SWA into account, it is unlikely that variations in NREMS intensity serve a thermoregulatory function.

It is concluded that in the rat, in accordance with the two-process model of sleep regulation, NREMS intensity, as indexed by SWA, is determined by the prior history of sleep and wakefulness. The present analyses further demonstrate that also the time course of T_{CRT} is to a large extent dependent on sleep and wakefulness. However this dependency of both sleep intensity and T_{CRT} on the vigilance state distribution is not associated with a mutual dependency of T_{CRT} and SWA.

SAMENVATTI

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